to get used to it, the stories of the miners in the hills undid him. Abandoning his office, to be proxied by T. M. Leavenworth, who finally succeeded him in October, 1848, he seized pick and shovel and was off to the mines. At the very moment when an intelligent, powerful hand might have saved the helpless infant city many a hard knock, Townsend turned from his duty to pursue a will-o'-the-wisp.

DOCTOR TOWNSEND'S SUBSEQUENT CAREER

In August he returned, little if any better off than when he left, and picked up his practice where he had let it drop. Such was his personality and his ability that he was elected a member of the Ayuntiamiento (town council) for 1849, and soon after that was chosen its president. In this capacity he was instrumental in electing the first city physician.

The mines had failed him, but he was not discouraged. As the population increased with such rapidity, so the value of city lots began to rise. He saw, as he looked to the south of the city, a great hill overlooking the south and east, with the southern arm of the bay at its doorstep. It was called the Potrero Nuevo and was inhabited chiefly by goats. He entered into a partnership with a Dutchman named Cornelius de Boom to subdivide this section and make out of it a pleasant suburban settlement. Unfortunately, even in 1849, and in spite of its delightful climate, the Potrero would not mold itself into a suburban locality, and the deal fell to pieces.

By this time Townsend had tried everything he could think of in the way of speculation—public office, mining, and medical practice. True to his philosophy, he blamed not himself but his environment for his trouble, and prepared to move once more. He sold his house on California Street to De Boom, bought a piece of 195 acres on the Milpitas Road near San Jose, and, murmuring about the injustices of an honest existence, grumpily moved thereto.

One matter cheered his dissatisfaction. After sixteen barren years of marriage, Elizabeth gave birth to a husky son. Then Townsend built an adobe house which he hoped would be the home for himself and his descendants for generations, and laid out a carefully chosen garden while the little boy crept and toddled among the iris and the new rose bushes.

THE FORTY-NINER DAYS

So passed 1849. The year 1850 opened with a roar, and was a year of savage contrasts. The emigrants suffered that year more acutely than ever. In January came torrential rains in California, and with the rains came bitter cold and hunger. The ill-equipped emigrants, now turned miners, in flapping tents and leaky cabins, had no food, no heat, and no money. To them came all the evils of this unhappy condition. Tuberculosis, dysentery, scurvy, influenza, and the various kindred ills of malnutrition, so depleted them that every available medical man was fully occupied in scratching the surface of their misery. From Sacramento to Monterey every doctor slaved to save what lives and help what pitiful wrecks he could until at last spring came, the weather changed, and life began again. The relief was not for long. Far out on the plain, creeping slowly but irresistibly closer, was a greater evil. Cholera, bred in the crowded cities of the east; rapacious, virulent, epidemic, creeping into train after train of wagons as it left on its adventure, was running like a prairie fire. Silently it rushed across the plain, leaving wailing survivors in its path. Silently it crept into the poor tents and cabins of the miners in the hills, and silently it reached the cities where it was met by its foul relative, cholera from Asia. Desperately the doctors worked against it, and as the summer waned so ceased the cholera. One final slap it gave with the back of its hand as it passed. In December, 1850, John Townsend lay dead of it, and so did Elizabeth, his wife. And when the relatives came into the house they found the little boy playing quietly but cheerfully, unharmed, on the floor beside his mother's body!

The old adobe is gone now and the son is gone, too, but the memory of the robust, pioneering father is as clear today as when, ninety years ago, he strode about the sandy streets of the tiny village by the bay.

University of California Medical Library.

CLINICAL NOTES AND CASE REPORTS

LOBAR PNEUMONIA DUE TO STREPTOCOCCI

USE OF BLOOD FROM AN IMMUNIZED DONOR IN ITS TREATMENT

By HENRY J. KRAAYMES, M. D.
Oakland

WITH the enormous advance in the diagnosis and treatment of lobar pneumonia, we still observe occasional types which cannot be expected to yield to typed serum administration. In the case history here presented, the patient had a lobar pneumonia, involving the right upper lobe and caused by type alpha and type gamma streptococci. There was obviously no point in administering antipneumococcal serum, and sulfanilamide was employed in the forlorn hope that it might act on the streptococci, although it was recognized that the chances were slim. While the patient was desperately ill, blood from a donor immunized against type alpha and type gamma streptococci was administered at the suggestion of Dr. A. P. Krueger, Professor of Bacteriology at the University of California. There was prompt improvement in the patient's status, leading to a rapid clinical recovery.

REPORT OF CASE

The patient was a business man, forty-eight years old, whose past history included pneumonia in 1918, amebic dysentery in the same year, and pneumonia again in 1930. He had a record of more than average indulgence in alcohol. The patient was first seen on February 3, 1939, when he came to the office for a general physical examination. In a routine x-ray examination the chest was found to be clear and the patient was pronounced in good general condition. On February 5, 1939, he complained of a sharp pain in the right parasternal region, becoming more severe upon deep inspiration. Physical examination revealed no abnormalities, although questioning elicited the fact that the patient had had a severe cold two weeks previously. On February 6 the patient developed a slight unproductive cough, he per-

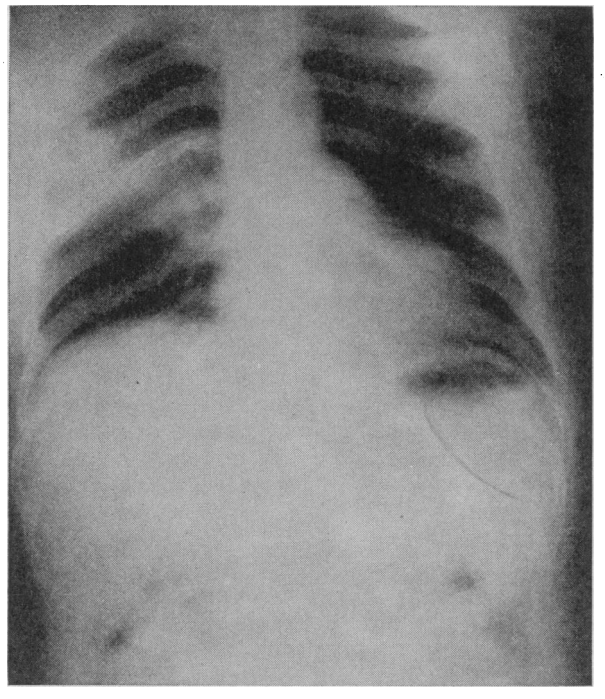

Fig. 1.—Bedside film of chest showing pneumonic infiltration in the right midlung.

spired freely and complained of persistent pain in the right parasternal region. His appearance was somewhat toxic, and examination revealed slight dullness on percussion over the right parasternal area in the region of the fourth and fifth rib, while a few moist râles were heard over this area. The temperature was 100.8 degrees, pulse 80, respiration 20, and it was deemed necessary to remove the patient to a hospital. His condition rapidly grew worse; he was decidedly toxic and was raising very little tough sputum containing no pneumococci, but very many short-chain streptococci. The white-cell count at this time was 20,500.

Intensive administration of neoprontosil was begun upon hospitalization. During the next few days the patient became very toxic, irrational, cyanotic, and dyspneic. The temperature rose to 103 degrees, pulse 114, and respiration 30. He was placed in an oxygen tent.

Pertinent *laboratory findings* were:

Sputum culture showed very many short streptococci of the alpha and gamma types, no beta hemolytic streptococci and no pneumococci. Repeated Neufeld typings were of no avail in the attempt to find pneumococci.

X-ray showed pneumonic infiltration in the right lung into what appears to be the periphery of the lower portion of the upper lobe. The density was very homogeneous in the periphery. The left lung field was clear.

After 120 cubic centimeters of 5 per cent neoprontosil had been given intramuscularly, the sulfanilamide therapy was continued with 60 grains of prontylin a day by mouth. However, the clinical picture became increasingly poor, the temperature remained high (103.4 by rectum), the pulse rate was 130 per minute, variable and of poor quality, respiration 36. The white count was 23,850 and blood culture was negative. Sulfanilamide therapy was discontinued because of the persistent cyanosis, severe headaches, and the blood findings. The red-cell count had dropped to 3,650,000 with 72 per cent hemoglobin; there was polychromasia and toxic granulation of the polymorphonuclear neutrophils.

On the advice of Doctor Krueger, the administration of citrated whole blood from a donor who had been immunized against type alpha and type gamma streptococci was undertaken. Three doses of 20 cubic centimeters each were given intramuscularly during the next twenty-four hours. After that one injection of 20 cubic centimeters was given every eighteen hours. Along with the usual pneumonia care the patient also received an ampoule of coramin every four hours, and 1000 cubic centimeters of 10 per cent glucose solution intravenously each day.

Within thirty-six hours the temperature began to drop and the patient became a little more rational; he was still very toxic and the quality of the pulse was poor. The white

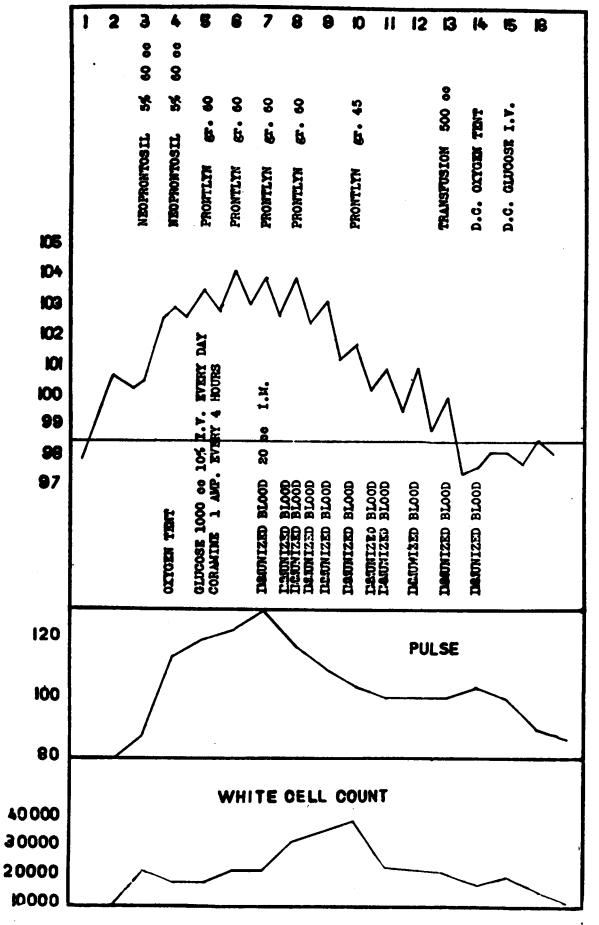

Chart 1.—Graphic chart showing the response of temperature, pulse and white cell count following the administration of immunized blood. Note, particularly, the prompt rise in white cell count.

cell count rose to 37,500, and x-ray examination revealed a slight increase in the pneumonic process in the right lung. Blood cultures remained consistently negative, and repeated sputum cultures continued to show large numbers of type alpha and type gamma streptococci.

On the eleventh day of hospitalization a transfusion of 500 cubic centimeters of normal blood was given by the indirect citrate method. Administration of immune blood in 20 cubic centimeters amounts intramuscularly every eighteen hours was continued, together with coramin and intravenous administration of glucose.

The patient improved greatly, and on the twelfth day of his illness he could be taken out of the oxygen tent for short periods without discomfort. On the thirteenth day of illness he received the eleventh and last injection of immune blood. The temperature was now 100 degrees; pulse 100, of fair quality; respiration, 22; and the white-cell count, 17,400. Further recovery was uneventful. X-ray examination of the chest before discharge from the hospital on the twenty-second day of illness showed the entire right lung field to be clear and fully aerated, with no evidence of fluid in either pleural cavity.

SUMMARY

A case of lobar pneumonia of a very toxic nature, involving only the right upper lobe, is reported. The total absence of pneumococci and the prevalence of type alpha and type gamma streptococci in the sputum cultures placed this type of pneumonia outside of the classification amenable to typed serum therapy. Sulfanilamide was given in the hope that it might be effective against the causal agents, although it was recognized that the chances were poor, since the organisms were of the nonhemolytic variety. Sulfanilamide seemed to have

no effect on the clinical picture and was discontinued because of severe toxic manifestations. Intramuscular administration of citrated whole blood from a donor who had been immunized against type alpha and type gamma streptococci was followed by a rapid improvement, and it is felt that the favorable outcome in this case must be ascribed to the effect of the immune blood. Doctor Krueger, from whom the blood was obtained, informs me that blood from donors (whom he immunizes against the various organisms by injecting undenatured bacterial antigens) has been used in the treatment of approximately five hundred cases of acute infection of various sorts. It is his opinion that the intramuscular injection of the blood establishes a depot in the tissues from which specific antibodies are absorbed, with resultant enhancement of the phagocytic activity of the reticuloendothelial system. Nearly always there is a prompt rise in the white-cell count (in this case from 23,000 to 40,000) and a diminution in toxemia. In general, the earlier the blood was administered the better have been the results.

400 Wakefield Building.

TRAUMATIC RUPTURE OF THE LIVER

REPORT OF CASE

By MAX E. PICKWORTH, M. D.
San Jose

RUPTURE of the liver is one of the rarely reported surgical emergencies. Most patients so afflicted die without benefit of surgery, and are included in the general group of "death due to shock." Shock is no contraindication to exploration; in fact, continued shock should be an indication to operate.

Hemorrhage is the outstanding danger of hepatic trauma. Control of that hemorrhage has to be the primary object of treatment. We know that spontaneous hemostasis of ruptured liver is rare. There have been numerous methods, combinations of methods, and modifications devised for hemostasis, and from this fact no one method probably can be applied to all cases. In these emergencies one has to use whatever is at hand. We must bear in mind that, if hemorrhage can be controlled for seventy-two hours, we have more than likely accomplished our purpose. The methods more commonly available for application, such as suturing of the liver with or without the additional support of strips from the abdominal wall or omentum and packing with gauze, are generally sufficient. However, most cases reported, in which packing and drainage were used, have had complications of abscess or secondary hemorrhage.

Adjuncts to the above would be compression of the portal vein, as employed by Pringle in 1908, for temporary control of hemorrhage. McDill, in 1912, clamped the vessels of the gastrohepatic omentum with an enterostomy clamp, and he states that procedures can thus probably be made entirely bloodless for eight to ten minutes with safety.

The liver's large size, its friability and its fixed position render it particularly vulnerable to external force. Anteroposterior compression is probably the most common cause of laceration.

According to Moynihan, subcutaneous wounds of the liver are of three kinds:

1. Rupture of liver with laceration of Glissons' capsule;
2. Separation of the capsule with subcapsular hemorrhage; and
3. Central rupture, leading to hematoma, and thence to abscess or cyst formation.

He further states that the right lobe is injured six times as often as the left.

When traumatized the liver has a tendency to split or crack in a stellate manner, with massive hemorrhage, and spilling of varying amounts of bile. These are the cases belonging to the first class, and the ones in which mortality is high unless operated within the first few hours. Those cases coming to operation several days following injury belong to the second and third classes.

Robertson and Graham report a case of subcapsular hemorrhage operated on twenty-seven days following injury. Christopher reports a case of primary subcapsular hemorrhage, with spontaneous rupture of the capsule on the operating table, within twenty-four hours of injury.

Frequently other abdominal viscera or thoracic organs and diaphragm are also injured, thus producing serious complications which may mask symptoms referable to the injured liver. Even in these cases much is to be hoped for by immediate and adequate surgery. This is borne out by the unusual and dramatic case reported by Gemmil and Martin. The patient, a woman of twenty-six, was injured by an automobile. There was evisceration of the intestine, severe laceration of the liver, and torn right kidney. The intestine was returned to the abdomen, and the liver and kidney were sutured, followed by an uneventful recovery in forty-six days.

Mortality figures for rupture of the liver run as high as 80 per cent in operated cases. Factors influencing mortality:

1. Acute anemia and shock.
2. Injuries to other viscera.
3. Paralytic ileus due to trauma or bile leakage.
4. Failure to estimate the gravity of the situation when first seen. Occasionally a trivial trauma may cause rupture, or severe hepatic injury may be accompanied by relatively insignificant primary symptoms, as in the case reported by Robin. A man fell across a ditch, striking his abdomen. He had only slight discomfort, walked to his car and was driven home. More than twelve hours later he was found to be bleeding severely from hepatic rupture. (This case may have been similar to the one reported by Christopher).
5. Failure to give the utmost attention to postoperative care.

The diagnosis of rupture of the liver may be difficult. A history of injury in the hepatic region always should make one suspect rupture of the liver. The differential diagnosis between visceral injury and simple shock or simple injury to the abdominal wall must be made. Repeated blood counts will aid greatly in this differentiation.

SYMPTOMS

1. Pain in right upper quadrant or generalized, with pain referred to right shoulder or back.